the gene or the part thereof is controlled by a promoter sequence, or an effective part thereof, and

ii) acetaminophen,

wherein the acetaminophen is converted in the cells into NABQI and wherein said cells do not express a sufficient level of glutathione to detoxify the NABQI.

IN THE ABSTRACT:

Please substitute the new Abstract of the Disclosure submitted herewith on a separate page for the original Abstract presently in the application.

REMARKS

The foregoing amendments are respectfully submitted to replace the claims presently on file. Applicants respectfully submit that the new claims and amended abstract are adequately supported by the originally filed specification.

Specifically, original claim 7 lists many examples of promoters, both tumor specific and constitutive. In addition, section 3.2 of the specification, especially at page 4, line 10 *et seq* explains that because the glutathione content of normal cells is higher than that of cancer cells, normal cells are able to withstand higher levels of NABQI. Accordingly, the original specification supports the method of using a vector comprising a P450 gene or its functional equivalent under the control of both tumor-specific and constitutive promoters. No new matter has been added, and entry and examination of the new claims on the merits are respectfully requested.

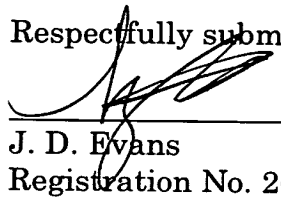
In addition, applicants have amended the paragraph bridging pages 9 and 10 of the specification, to correct a typographical error. Specifically, "zilin gene promoter" is replaced with "villin gene promoter." One of skill in the art would recognize readily that there is no such gene as 'zilin,' while in contrast the villin gene is well-known and well characterized and its promoter is routinely used as a strong constitutive promoter.

If there are any questions regarding this amendment or the application in general, a telephone call to the undersigned would be appreciated since this should expedite the prosecution of the application for all concerned.

If necessary to effect a timely response, this paper should be considered as a petition for an Extension of Time sufficient to effect a timely response, and please charge any deficiency in fees or credit any overpayments to Deposit Account No. 05-1323 (Docket #2190/49927).

November 14, 2002

Respectfully submitted,



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VERSION WITH MARKINGS SHOWING CHANGES MADE

IN THE SPECIFICATION:

The paragraph bridging pages 9 and 10 has been amended as follows:

In a still further preferred method of the invention said promoter sequence is preferably selected from at least one of the following: TRP-1; HER2; HER3; ERBB2; ERBB3; CEA; MUC-1; α-fetoprotein; Rous sarcoma virus long terminal repeat; cytomegalovirus promoter; murine leukaemia long terminal repeat; simian virus 40 early and late promoters; herpes simplex virus thymidine kinase promoter; prostate specific antigen promoter (PSA); [zilin] villin gene promoter; pancreatic amylase promoter; tyrosinase related peptide promoter; tumour rejection antigen precursor promoters.

IN THE ABSTRACT:

A therapeutic method [of cancer therapy] which exploits the cytotoxic properties of acetaminophen when converted to NABQI by the metabolic activity of [tumor cell specific] P450; vectors for delivering P450 to tumor cells, and therapeutic compositions comprising such vectors.

ABSTRACT OF THE DISCLOSURE

A therapeutic method which exploits the cytotoxic properties of acetaminophen when converted to NABQI by the metabolic activity of P450; vectors for delivering P450 to tumor cells, and therapeutic compositions comprising such vectors.